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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.
08/997,464	12/23/97	STERN D	54202/JPW/SB

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EXAMINER	
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ART UNIT	**PAPER NUMBER**
1633	11

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Please find below and/or attached an Office communication concerning this application or proceeding.

Commissioner of Patents and Trademarks

Office Action Summary	Application No. 08/997,464	Applicant(s) Stern et al.
	Examiner Janet M. Kerr	Group Art Unit 1633

☒ Responsive to communication(s) filed on *Jul 3, 2000*.

☒ This action is **FINAL**.

☐ Since this application is in condition for allowance except for formal matters, **prosecution as to the merits is closed** in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11; 453 O.G. 213.

A shortened statutory period for response to this action is set to expire *3* month(s), or thirty days, whichever is longer, from the mailing date of this communication. Failure to respond within the period for response will cause the application to become abandoned. (35 U.S.C. § 133). Extensions of time may be obtained under the provisions of 37 CFR 1.136(a).

Disposition of Claims

☒ Claim(s) *1-5, 11, 12, and 34-37* is/are pending in the application.

Of the above, claim(s) _____ is/are withdrawn from consideration.

☐ Claim(s) _____ is/are allowed.

☒ Claim(s) *1-5, 11, 12, and 34-37* is/are rejected.

☐ Claim(s) _____ is/are objected to.

☐ Claims _____ are subject to restriction or election requirement.

Application Papers

☐ See the attached Notice of Draftsperson's Patent Drawing Review, PTO-948.

☐ The drawing(s) filed on _____ is/are objected to by the Examiner.

☐ The proposed drawing correction, filed on _____ is ☐approved ☐disapproved.

☐ The specification is objected to by the Examiner.

☐ The oath or declaration is objected to by the Examiner.

Priority under 35 U.S.C. § 119

☐ Acknowledgement is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d).

☐ All ☐ Some* ☐ None of the CERTIFIED copies of the priority documents have been

☐ received.

☐ received in Application No. (Series Code/Serial Number) _____.

☐ received in this national stage application from the International Bureau (PCT Rule 17.2(a)).

*Certified copies not received: _____.

☐ Acknowledgement is made of a claim for domestic priority under 35 U.S.C. § 119(e).

Attachment(s)

☒ Notice of References Cited, PTO-892

☐ Information Disclosure Statement(s), PTO-1449, Paper No(s). _____

☐ Interview Summary, PTO-413

☐ Notice of Draftsperson's Patent Drawing Review, PTO-948

☐ Notice of Informal Patent Application, PTO-152

— SEE OFFICE ACTION ON THE FOLLOWING PAGES —

Response to Amendment

Applicants' amendment, filed on 10/18/99, has been entered.

Claims 6-10 and 13-33 have been canceled.

Claims 34-37 have been added.

Claims 1-5, 11, 12, and 34-37 remain pending.

Claim Rejections - 35 USC § 112

The following is a quotation of the second paragraph of 35 U.S.C. 112:

> The specification shall conclude with one or more claims particularly pointing out and distinctly claiming the subject matter which the applicant regards as his invention.

Claims 1-5, 11, 12, and 34-37 are/remain rejected under 35 U.S.C. 112, second paragraph, as being indefinite for failing to particularly point out and distinctly claim the subject matter which applicant regards as the invention for the reasons of record and the reasons below.

Claim 1 is vague and indefinite for the following reasons: it is unclear which cell types are suitable for evaluating neurotoxicity; it is unclear if the transfected DNA is actually expressed in the cell; and it is unclear what concentration of amyloid-beta peptide is required and which amyloid-beta peptide is required as there is no indication in the claim as to the function of the amyloid-beta peptide in the method. Thus, it is unclear which peptide in what amount is required to practice the method.

Claim 4 is rendered vague and indefinite by the phrase "a solid support" as there is no definition or examples of solid supports in the specification. Thus, it is unclear what is encompassed by "a solid support".

Claims 36 and 37 are rendered vague and indefinite by the phrase "encodes for" as it is unclear how a DNA can "encode for" a protein. It is suggested that the phrase be amended to "encodes".

Applicant's arguments filed 7/3/00 have been fully considered but they are not persuasive. Applicants argue that with regard to "solid support", the plain language of this claim renders the claim language definite. Applicants assert that a solid support is known to one of skill in the art as a material to which a compound can be affixed. Applicants provide Exhibits A-C which are references describing solid supports, published prior to the subject application's filing date. Applicants argue that the documents make clear that one of skill in the art would know the term solid support, and therefore this term is not vague. This argument is not persuasive. While "solid supports" per se are well known in the art, as evidenced in Exhibits A-C the specification does not provide any definition of the type of solid support which could be used in the method. The solid supports described in Exhibits A-C are not utilized in cell culture systems which is required in the claim. Thus, when interpreted in light of the specification, and given the absence of any definition of "solid support" or working example of "solid support" in the method, it is not clear what type of "solid support" should be used in the method.

The following is a quotation of the first paragraph of 35 U.S.C. 112:

> The specification shall contain a written description of the invention, and of the manner and process of making and using it, in such full, clear, concise, and exact terms as to enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the same and shall set forth the best mode contemplated by the inventor of carrying out his invention.

Claims 3, 4, 11, and 12 remain rejected under 35 U.S.C. 112, first paragraph, because the specification, while being enabling for a method for evaluating the ability of a compound to inhibit neurotoxicity comprising contacting a neuronal cell or a neuronally differentiated PC12 cell with a compound, and a pharmaceutical composition for use, *in vitro*, comprising a compound identified by the method, does not reasonably provide enablement for a method for evaluating the ability of a compound to inhibit neurotoxicity wherein the compound is a peptidomimetic, wherein the compound is bound to a solid support, or a pharmaceutical composition for use, *in vivo*. The

specification does not enable any person skilled in the art to which it pertains, or with which it is most nearly connected, to make and use the invention commensurate in scope with these claims.

The claimed invention is directed to a method for evaluating the ability of a compound to inhibit neurotoxicity. In claim 3, the compound is a peptidomimetic. In claim 4, the compound is bound to a solid support. Claims 11 and 12 are directed to a pharmaceutical composition comprising the compound identified in claim 1.

As stated in the office action of 1/3/00, with regard to providing a compound which is a peptidomimetic, the specification does not define what would encompass a suitable peptidomimetic, nor does the specification identify any particular peptidomimetic or method for selecting a peptidomimetic which is suitable for use in the method for evaluating neurotoxicity. Absent any guidance in the specification for selecting peptidomimetics, one of skill in the art would not have a high expectation of successfully isolating and utilizing a peptidomimetic in the claimed method without undue experimentation.

Applicant's arguments filed 7/3/00 have been fully considered but they are not persuasive. Applicants argue that peptidomimetics would have been fully known to one of ordinary skill in the at the time of filing, and refers to U.S. Patent No. 5,612,895, issued March 18, 1997, which discusses the making of peptidomimetics. Applicants further argue that there are many possible "suitable" peptidomimetics which would be encompassed by the claimed invention. These arguments are not persuasive as neither the specification nor applicants' arguments provide any particular example of a suitable peptidomimetic, nor does the specification identify any particular compound in which a peptidomimetic could be derived based on the teachings of U.S. Patent No. 5,612,895. In view of the lack of guidance in the specification as to any particular compound which is suitable in the assay, and therefore any peptidomimetic which can be derived from the compound, one of skill in the art would not have been able to make or use the peptidomimetic in the claimed method.

As stated in the office action of 1/3/00, with regard to utilizing a compound bound to a solid support, the specification does not define what is intended by a solid support nor does the specification disclose any solid supports which are suitable for use in the claimed method. In addition, the specification does not teach the structures or classes of compounds which may be bound to solid supports. As the specification does not disclose solid supports or compounds which are suitable for attachment to a solid support, one of ordinary skill in the art would not know which supports to use for which compounds, nor would one of skill in the art know if any and all compounds are actually capable of binding to solid supports. Thus, one of skill in the art would not have had a high expectation of successfully ascertaining which particular compound should be bound to a particular solid support without undue experimentation.

Applicant's arguments filed 7/3/00 have been fully considered but they are not persuasive. Applicants argue that with regard to "solid support", the plain language of the claim and the specification is sufficient to enable the claimed invention. Applicants assert that a solid support would have been known to one of ordinary skill in the art, and further that a particular solid support would be known be known by one of ordinary skill to be suitable for a particular type of compound. Applicants indicate, as examples, that it is known that certain compounds bond well to silica type materials, other compounds bind well to plastics, and still other materials bind well to metals. These arguments are not persuasive as the specification does not provide guidance as to the type of compound which is added to the culture such that a suitable solid support, which binds to the compound can be selected. Moreover, there is no indication how the compound, bound to the solid support, is added to the culture system. While solid supports and specific compounds which bind to these supports may be well established in the art as asserted by applicants, there is no teaching in the specification, nor is there any example provided by applicants' arguments which indicate which compound and appropriate solid support to select, and how to add the compound bound to the solid support to the culture system. The specification does not provide any guidance as to how to make or use the compound/solid support element of the claimed invention.

Claims 11 and 12 are directed to a pharmaceutical composition, wherein the composition comprises a compound which inhibits neurotoxicity and a pharmaceutically acceptable carrier. As written, the pharmaceutical composition encompasses both *in vitro* and *in vivo* applications. Moreover, the compound can encompass macromolecules such as nucleic acids.

While the specification is enabling for providing a pharmaceutical composition to cells *in vitro* to establish whether the compound inhibits neurotoxicity, the specification is non-enabling for administering a pharmaceutical composition which inhibits neurotoxicity *in vivo*. The specification does not provide any correlation with respect to the *in vitro* and *in vivo* effectiveness of a compound as the inhibition of neurotoxicity has only been demonstrated *in vitro*.

As indicated in the office action of 1/3/00, the specification does not disclose which compound would be suitable for treating a particular disease, whether the same compound would successfully treat all recited diseases, what dosages and routes of administration of the compound would be effective in treating a specific disclosed disease, and how one would ascertain whether the pharmaceutical composition was effective in ameliorating a specific disclosed disease. In addition, the state of the art at the time of filing indicates that treating neurodegenerative diseases is neither routine nor predictable.

Applicant's arguments filed 7/3/00 have been fully considered but they are not persuasive. Applicants argue that the examiner has not provided any support for this position, and assert that administration of compounds which would inhibit neurotoxicity would have been well known to one of ordinary skill in the art. Applicants refer to Exhibits D and F to support the arguments that providing a pharmaceutical composition of a neurotoxicity inhibitor for administration is well known in the art. Moreover, applicants rely on Carter-Wallace, Inc. v Davis-Edwards Pharmacal Corp. To indicate that the courts have recognized that determining an effective dosage for a pharmaceutical agent against a particular disease indication is well within the ordinary skill of the art. These arguments are not persuasive. Exhibit D teaches administration of a specific, well characterized pharmaceutical, cyclosporin A, to a subject. Contrary to the teachings in Exhibit D, the specification does not provide any compound which is characterized such that one of skill in

the art would be able to determine its mechanism of action, *in vivo* turnover rates, the mode of administration, the amount to administer and the frequency of administration. Exhibit F is a table of contents from "Biological Approaches to the Controlled Delivery of Drugs". A table of contents does not provide any information, aside from a title of an article and the authorship, by which one of skill in the art would be able to ascertain how to provide and administer an undisclosed compound in a pharmaceutical composition.

With regard to the Sabate *et al.* reference which was relied upon in the office action of 1/3/00 to provide evidence that treating neurodegenerative diseases is neither routine nor predictable, applicants argue that the blood brain barrier is merely one challenge in optimizing administration of an inhibitor of neurotoxicity, e.g., intracranial implants or injections are possible modes of administration which could be successful. This argument is not persuasive as applicants are providing hypothetical examples of **possible** modes which **could be** successful, which are not described in the specification. Applicants also point out that there are no claims pending to "treatment of neurodegenerative diseases", however, the pharmaceutical composition recited in the claims are fully enabled by the subject specification. While the examiner agrees that there are no treatment method claims, as indicated in the office action of 1/3/00, the intended use of the pharmaceutical composition is for treatment of a myriad of neurodegenerative diseases and disorders. As the specification does not provide any example of a compound obtained by a process which could be incorporated into a pharmaceutical composition and which can be used in a treatment method for any one of the disclosed neurodegenerative diseases or disorders, one of skill in the art would not be able to make or use the pharmaceutical composition as intended in a predictable and reproducible manner and without undue experimentation. Thus, while the specification is enabling for a pharmaceutical composition which can be used in *in vitro* applications, the specification is not enabling for a pharmaceutical composition which can be used in *in vivo* applications.

With regard to applicants argument that the method is enabled for use in vivo, applicants assert that the cells transfected with DNA encoding RAGE and mutant PS-2 could be part of the

organism and that the claimed method could be carried out in vivo by routine methods. Applicants suggest that one of ordinary skill in the art could make a transgenic mouse which has been engineered with a DNA construct which encodes RAGE and a mutant PS-2 and the mouse could be administered a compound and amyloid-beta peptide, and subsequently analyzed for apoptotic activity. This argument is not persuasive as the specification does not disclose how to make transgenic mice comprising a heterologous RAGE and a mutant PS-2, nor does the specification teach a particular phenotype displayed by the transgenic mice. Moreover, the generation of transgenic mice which display a particular phenotype is neither routine nor predictable. For example, Palmiter *et al.* (Proc. Natl. Acad. Sci, USA, 1991) teach that directed expression of any gene to any specific cell type of an animal by using established transgenic methodology is theoretically possible by combining the regulatory region(s) of a gene that is expressed in a cell-specific manner with any mRNA-encoding structural gene. Palmiter *et al.* note, however, that not all gene constructs work well; the two most common problems are inappropriate expression patterns and failure to achieve adequate expression levels (see page 478, left column, first paragraph). Kappel *et al.* (Current Opinion in Biotechnology, 3:548-553, 1992) teach that while transgenes can be targeted, inherent cellular mechanisms may alter the pattern of gene expression (see, e.g., page 549, right column). In addition, Cameron (Molecular Biology, 7:253-265, 1997) teaches that

"Well-regulated transgene expression is the key to successful transgenic work, but all too often experiments are blighted by poor levels or the complete absence of expression, as well as less common problems, such as leaky expression in nontargeted tissues....". "A feature common to many transgenic experiments is the unpredictable nature of transgene expression with different transgenic lines produced with the same construct frequently displaying different levels of expression. Further, expression levels do not correlate with the number of transgene copies integrated...". "Such copy-number-independent, integration-site-dependent expression patterns emphasize the influence of surrounding chromatin on the transgene" (see, e.g., page 256 under "Transgene Regulation and Expression").

As the specification does not disclose how to make such a transgenic mouse, and view of the unpredictability of generating a transgenic mouse with a particular phenotype as indicated in

the state of the art of transgenics, applicants reliance on a hypothetical example of providing the pharmaceutical composition to a non-disclosed transgenic mouse is not persuasive.

Applicants argue that the degree of experimentation needed to practice the claimed invention is well within the amount of experimentation that is willingly and routinely undertaken by those of ordinary skill in the relevant arts. Applicants allege that such experimentation, which is guided by applicants' disclosure viewed in light of earlier studies, is very clearly not undue. Applicants rely on In re Wands to support the position that the experimentation required is not undue given the nature of the claimed invention and the state of the art. This is not persuasive as the specification fails to provide sufficient guidance with regard to providing peptidomimetics or solid supports to be used in the assay, and fails to provide sufficient guidance as to what the pharmaceutical composition comprises and how to administer the pharmaceutical composition given the disclosure of the intended use of such a pharmaceutical composition in the instant application.

Claim Rejections - 35 USC § 102

The following is a quotation of the appropriate paragraphs of 35 U.S.C. 102 that form the basis for the rejections under this section made in this Office action:

> A person shall be entitled to a patent unless --
>
> (b) the invention was patented or described in a printed publication in this or a foreign country or in public use or on sale in this country, more than one year prior to the date of application for patent in the United States.

Claims 11 and 12 remain rejected under 35 U.S.C. 102(b) as being anticipated by Bartus *et al.* (U.S. Patent No. 5,444,042, 1995) for the reasons of record, and the reasons below.

The claims are directed to a pharmaceutical composition comprising a compound capable of inhibiting neurotoxicity, claimed in a product-by-process format.

As indicated in the office action of 1/3/00, Bartus *et al.* teach compounds which inhibit neurotoxicity, i.e., calpain inhibitors. The calpain inhibitors effectively block cell death in an *in*

vitro model for neuropathology (see column 73, lines 5-24). The compounds can be formulated as pharmaceutical compositions comprising the compound of interest in a pharmaceutically acceptable formulation containing a carrier material (see column 4, lines 48-54 and column 66, lines 36-40).

Applicants argue that the reference does not anticipate the claimed invention in that Bartus *et al.* do not teach that the product was identified by the process steps of the claims. However, as indicated in MPEP 2113:

"Even though product - by process claims are limited by and defined by the process, determination of patentability is based on the product itself. The patentability of a product does not depend on its method of production. If the product in the product - by - process claim is the same as or obvious from a product of the prior art, the claim is unpatentable even though the prior product was made by a different process." In re Thorpe , 227 USPQ 964, 966 (Fed. Cir. 1985).

The claimed pharmaceutical composition is obtained by a particular process. However, the process does not provide any structural characteristics of the composition. The only functional limitation of the composition is that inhibits neurotoxicity. In this regard, the pharmaceutical composition of Bartus *et al.* also inhibits neurotoxicity. Thus, the neurotoxicity inhibitor of Bartus *et al.* anticipates the claimed neurotoxicity inhibitor.

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to

the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(f) or (g) prior art under 35 U.S.C. 103(a).

Claims 1-5, 11, 12, and 34-37 are rejected under 35 U.S.C. 103(a) as being unpatentable over Wolozin *et al.* (Science, 274:1710-1713, December 6, 1996) taken with Yan *et al.* (Nature, 382:685-691, 1996, newly applied).

Applicant's arguments with respect to the claims have been considered but are moot in view of the new ground(s) of rejection.

The claimed invention is drawn to a method of evaluating the ability of a compound to inhibit neurotoxicity and pharmaceutical compositions comprising compounds identified by the method.

Wolozin *et al.* disclose that transfecting neuronally differentiated PC12 cells with a mutant presenilin-2 protein (e.g., N141I) causes increased basal apoptosis in nerve growth factor-differentiated PC12 cells (see, e.g., page 1711, left column, middle column, and Figure 1). In addition, Wolozin *et al.* disclose a method comprising a) culturing the neuronally differentiated PC12 cells in the presence or absence of a compound, i.e., pertussis toxin or Aβ(1-42), b) determining the level of apoptosis in the control and treated cells, and c) comparing the extent of apoptotic activity in the cells cultured in the presence of the compound compared to cells cultured in the absence of the compound to evaluate the effect of the compound on apoptotic activity (see, e.g., page 1711, middle and right columns, page 1712, left column, Figure 3, and Figure 4E). The Aβ(1-42) compound is added to the cells at a concentration of 10 μM and was generated from a 1 mM Aβ(1-42) stock solution (see, e.g., page 1713, Note #21). Thus, Wolozin *et al.* disclose the claimed method and pharmaceutical composition comprising a compound and a pharmaceutical carrier. While Wolozin *et al.* do not disclose adding a nucleic acid compound to neuronally differentiated PC12 cells expressing a mutant presenilin-2 protein, or all of the claim-designated pharmaceutical carriers, Wolozin *et al.* does disclose adding PS-2 or ALG-3 antisense nucleic

acids to neuronally differentiated PC12 cells which do not express a mutant presenilin-2 protein. Addition of the antisense nucleic acid results in a decrease in apoptotic activity in the PC12 cells (see, e.g., page 1720, middle and right columns, and Figure 1). Inasmuch as Wolozin *et al.* disclose that PC12 cells that express a mutant presenilin-2 protein have a high apoptotic activity, it would have been obvious to add PS-2 or ALG-3 antisense nucleic acids to neuronally differentiated PC12 cells expressing mutant presenilin-2 to determine if the antisense nucleic acids are effective in decreasing the observed apoptotic activity in PC12 cells expressing mutant presenilin-2 protein. Moreover, adding the nucleic acids, or other compounds such as pertussis toxin or Aβ(1-42) to cell cultures as a pharmaceutical composition would have been obvious and well within the purview of one of ordinary skill in the art of cell culture. One of ordinary skill in the art would have been motivated to admix the compound of interest with a suitable carrier to more easily control the concentration of the compound added to the cell culture and to avoid a localized high concentration of a solid compound which may be detrimental to the cells.

Wolozin *et al.* do not teach that the PC12 cells are transfected with a DNA sequence encoding RAGE and which is expressed in PC12 cells. However, Yan *et al.* teach that enhanced expression of RAGE in Alzheimer's diseases, in affected neurons, in microglia and in vasculature, is consisting with the concept that Aβ-RAGE interaction may contribute to neurotoxicity that results in dementia (see page 382, left column, last paragraph). Thus, it would have been obvious to one of ordinary skill in the art to provide cells associated with neurodegenerative diseases, in a method of identifying compounds which inhibit neurotoxicities associated with neurodegenerative diseases. Yan *et al.* further teach that human Aβ(1-40 or Aβ(1-42) purified from plaques or vascular amyloid from Alzheimer's disease patients inhibits binding of Aβ to RAGE (see, e.g., page 688, left column); that Aβ binding to RAGE and Aβ-induced cellular perturbation results in oxidant stress and cytotoxicity (see, e.g., page 688, right column, under "RAGE and Aβ-induced cellular stress"). Yan *et al.* indicate that RAGE can mediate Aβ-induced oxidant stress on endothelium and neuronal cells and that the stress can be prevented by blocking access to RAGE using either anti-RAGE IgG or excess soluble receptor, and further teach that expression of

RAGE increases vulnerability to Aβ. Yan *et al.* indicate that RAGE, if present and/or upregulated in cells important in the pathogenesis of Alzheimer's disease, could mediate toxic effects when associated with Aβ. Note also that Yan *et al.* teach transfection of RAGE into COS-1 cells and the use of these transfected cells in analyzing the effect of compounds on Aβ activity with respect to oxidant stress (see, e.g., page 688, under "RAGE and Aβ-induced cellular stress").

It would have been obvious to one of ordinary skill in the art at the time the claimed invention was made to modify the method of Wolozin *et al.* by further modifying the presenilin-2 transfected PC12 cells of Wolozin *et al.* by transfecting the cells with a vector encoding RAGE in view of the teachings of Yan *et al.* that cells transfected with RAGE are useful in studying the interaction of RAGE and Aβ on oxidant stress and cytotoxicity in cells. One of skill in the art would have been motivated to provide such a modified PC12 cell to use in a method of identifying inhibitors of neurotoxic compounds, such as those associated with Alzheimer's disease, in view of the teachings of Yan *et al.* that enhanced expression of RAGE in Alzheimer's disease, in affected neurons, in microglia and in vasculature, is consisting with the concept that Aβ-RAGE interaction may contribute to neurotoxicity that results in dementia.

Thus the claimed invention as a whole was clearly *prima facie* obvious at the time the claimed invention was made especially in the absence of sufficient, clear, and convincing evidence to the contrary.

No claims are allowed.

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL.** See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for response to this final action is set to expire THREE MONTHS from the date of this action. In the event a first response is filed within TWO MONTHS of the mailing date of this final action and the advisory action is not mailed until after the end of the THREE-MONTH shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event will the statutory period for response expire later than SIX MONTHS from the date of this final action.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Janet M. Kerr whose telephone number is (703) 305-4055. Should the examiner be unavailable, inquiries should be directed to John LeGuyader, Supervisory Primary Examiner of Art Unit 1633, at (703) 308-0447. Papers related to this application may be submitted to Group 1600 by facsimile transmission. Papers should be faxed to Group 1600 via the PTO Fax Center located in Crystal Mall 1. The faxing of such papers must conform with the notice published in the Official Gazette, 1096 OG 30 (November 15, 1989). The CM1 Fax Center number is (703) 305-7401. Any inquiry of a general nature or relating to the status of this application should be directed to the Group 1600 receptionist whose telephone number is (703) 308-0196.

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